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What aspects of face processing are impaired in developmental prosopagnosia?

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Abstract

Developmental prosopagnosia (DP) is a severe impairment in identifying faces that is present from early in life and that occurs despite no apparent brain damage and intact visual and intellectual function. Here, we investigated what aspects of face processing are impaired/spared in developmental prosopagnosia by examining a relatively large group of individuals with DP ($n = 8$) using an extensive battery of well-established tasks. The tasks included measures of sensitivity to global motion and to global form, detection that a stimulus is a face, determination of its sex, holistic face processing, processing of face identity based on features, contour, and the spacing of features, and judgments of attractiveness. The DP cases showed normal sensitivity to global motion and global form and performed normally on our tests of face detection and holistic processing. On the other tasks, many DP cases were impaired but there was no systematic pattern. At least half showed deficits in processing of facial identity based on either the outer contour or spacing of the internal features, and/or on judgments of attractiveness. Three of the eight were impaired in processing facial identify based on the shape of internal features. The results show that DP is a heterogeneous condition and that impairment in recognizing faces cannot be predicted by poor performance on any one measure of face processing.

Keywords: Prosopagnosia; Face processing; Global motion; Global form; Face detection; Holistic processing; Configural processing; Featural processing; Attractiveness

1. Introduction

Adults are 'experts' in face processing: they can recognize thousands of individual faces rapidly and accurately, and they can easily decipher various cues, such as sex of face, emotional expression, and direction of gaze (see Bruce & Young, 1998, for a review). This proficiency in face recognition is remarkable considering that all human faces share the same basic arrangement of features (two eyes above a nose, that is above a mouth), and those features are highly similar in all individuals. While most adults are experts in face recognition (Carey, 1992), there exist rare cases of individuals who are severely impaired in face recognition, a clinical condition known as prosopagnosia. Documenting the pattern of their deficits may increase our understanding of the developmental processes underlying normal face perception.

Most studies have involved individuals who acquired prosopagnosia (AP) after damage to occipital-temporal cortex (e.g., Damasio, Damasio, & van Hoessen, 1982; Sergent & Villemure, 1989). However, there exist individuals that

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have impairment in face recognition all their lives despite no known brain injury. The term developmental prosopagnosia[1] (DP) refers to the absence of any known lesion or neurological condition that could account for the impairment in face recognition, and excludes individuals suffering from visual deprivation, such as congenital cataract, or developmental problems such as autism spectrum disorder. While interest in DP continues to grow, current knowledge of this condition is limited and in general the findings have been contradictory and inconsistent. This may be due to the small number of reported cases, the heterogeneity of the condition, the prevalence of single case studies, and/or the variability in the methods used to examine DP (for reviews see Behrmann & Avidan, 2005; Kress & Daum, 2003a).

Previous studies of individuals with DP typically have involved a single case and a limited number of tasks (Ariel & Sadeh, 1996; Bentin, Deouell, & Soroker, 1999; de Gelder & Rouw, 2000a; Duchaine, 2000; Duchaine, Nieminen-von Wendt, New, & Kulomaki, 2003; Duchaine, Parker, & Nakayama, 2003; Jones & Tranel, 2001; McConachie, 1976; Nunn, Postma, & Pearson, 2001; but see Behrmann, Avidan, Marotta, & Kimchi, 2005, for a more systematic study of 5 cases). These studies have indicated that there is variability in performance across tasks and across individuals with DP. Of course, all DP cases have trouble with facial identity, but tests with familiar faces (celebrities and acquaintances) have shown that some individuals with DP can recognize faces after a large number of exposures (Duchaine et al., 2003; Nunn et al., 2001) whereas others have trouble even with commonly seen faces (Barton, Cherkasova, Press, Intriligator, & O'Connor, 2003; Duchaine, 2000; Duchaine & Nieminen-von Wendt et al., 2003). The use of standardized clinical tests of face recognition, such as the Warrington Recognition Memory for Faces (RMF; Warrington, 1984) and the Benton Facial Recognition Test (BFRT; Benton, Sivan, Hamsher, Varney, & Spreen, 1983), have also revealed inconsistent findings. While some individuals with DP show deficits on these standardized tests (e.g., Ariel & Sadeh, 1996; de Gelder & Rouw, 2000a), others perform within the normal range despite clear impairment on tests of familiar face recognition (e.g., Duchaine, 2000; Nunn et al., 2001). The validity of these standardized measures has been criticized because the photos used in testing contain non-facial cues such as hairstyle and clothing (Duchaine & Weidenfeld, 2003; Kress & Daum, 2003a). In fact, on modified versions of the RMF and BFRT in which facial cues are removed by occluding the inner portion of the test faces, the accuracy of both normal controls and developmental prosopagnosics alike is within the normal range (Duchaine & Nakayama, 2004; Duchaine & Weidenfeld, 2003). Thus, normal performance on the BFRT and RMF by prosopagnosic individuals should be interpreted with caution, especially when reaction time measures are absent (see Delvenne, Seron, Coyette, & Rossion, 2004).

Investigations into the neural bases of DP also have found inconsistencies. Structural studies usually report no obvious abnormalities (Duchaine & Nieminen-von Wendt et al., 2003; Kress & Daum, 2003b; Nunn et al., 2001), but one case (YT) had a significantly smaller right temporal lobe compared to normals (Bentin et al., 1999). Some cases of DP show an abnormally small difference in the ERP response to faces versus objects for the 'N170', which is normally characterized by much greater negativity occurring 170 ms after stimulus onset for faces than for a variety of non-face object categories (Bentin, Allison, Puce, Perez, & McCarthy, 1996; Bentin et al., 1999; Kress & Daum, 2003b). In other cases, the N170 is not modulated normally by the inversion of the face or its presentation in the left temporal versus nasal visual field (de Gelder & Stekelenburg, 2005). Most cases of DP who have undergone fMRI have shown normal activation of the 'fusiform face area' or FFA (Avidan, Hasson, Malach, & Behrmann, 2005; Hasson, Avidan, Deouell, Bentin, & Malach, 2003), a region in the occipito-temporal cortex that responds more to faces than to most other stimulus categories (Kanwisher, McDermott, & Chun, 1997; McCarthy, Puce, Gore, & Allison, 1997). Yet an apparently normal FFA in a prosopagnosic may nevertheless show inefficient interactions with working memory and attention (DeGutis, Sagiv, D'Esposito, & Robertson, 2004). There are also three documented cases of DP without selective activation for faces within the FFA (Hadjikhani & de Gelder, 2002).

Individuals with DP often have impairments with other aspects of face processing, but again some individuals have shown normal abilities while others are impaired. This is true for recognition of facial expressions of emotion (Ariel & Sadeh, 1996; de Haan & Campbell, 1991; Duchaine et al., 2003; Jones & Tranel, 2001; McConachie, 1976; Nunn et al., 2001), and gender discrimination (Ariel & Sadeh, 1996; de Haan & Campbell, 1991; Jones & Tranel, 2001; Nunn et al., 2001). In most cases non-face object processing is intact, and when deficits in object recognition are present they are much less pronounced than face processing impairments (Ariel & Sadeh, 1996; Barton et al., 2003; Behrmann et al., 2005; Bentin et al., 1999; de Haan & Campbell, 1991; Duchaine & Nakayama, 2005; Nunn et al., 2001). In addition, a number of DP cases have severe impairments with navigation (Duchaine et al., 2003), suffer from auditory processing deficits (Duchaine, 2000; McConachie, 1976; Temple, 1992), and show interference between local elements and global shape under conditions in which global shape is dominant in normal controls, as if local details dominate their processing of objects (Behrmann et al., 2005). While there is no conclusive

[1] The terms "congenital prosopagnosia" and "developmental prosopagnosia" have been used interchangeably to refer to a condition involving a severe deficit in face processing in the absence of any observable cortical damage. However, congenital prosopagnosia has the added implication that the deficit was present from birth. While the participants in the present study have no evidence of cortical damage, recall no incident such as meningitis or accident that could have caused the impairment, and remember problems with face recognition all their lives, there is no way to ascertain whether their face processing impairment was in fact present at birth. To be conservative, we refer to these individuals as having developmental prosopagnosia (DP).